ilium. Strangely enough Stierlin's sign is absent in cancer of the caecum. Patient R. D., 29 years old, tailor. Father died of tuberculosis. Patient came to the University of California service, San Francisco Hospital, suffering from pain in the right iliac region. He had been treated for pulmonary tuberculosis which was of about one year's duration. His present illness began with a sudden pain in the right lower quadrant which disappeared in a few minutes some two and one-half weeks before admission to the hospital. This pain recurred suddenly while walking and was accompanied by nausea and vomiting. The pain spread to the whole abdomen. The patient was admitted to the hospital with a fever of 100.5°. Examination of the abdomen showed slight rigidity of the entire abdomen, lower more than the upper, and of the right more than the left, considerable tenderness in the outer half of the right iliac fossa. An elongated round boggy mass could be palpated at McBurney's point, which was very tender. With rest in bed the pain and local symptoms disappeared in the course of a week so that there remains at the present time a patient with pulmonary tuberculosis and a mass about 2½ inches long by an inch wide, sausage-shaped, lying obliquely in the region of the appendix only slightly tender on pressure and which undoubtedly is a hypertrophic tuberculoma either of the ilium or of the caecum. Stierlin's sign being present in this case we take it to be a lesion of the ilium.

Chronic hyperplastic tuberculosis while probably more common in the iliocæcal region has also been observed in the sigmoid flexure. The diagnosis of subserous fibroma should be avoided until tuberculosis has been excluded.

In conclusion it may be said that tuberculosis of the abdominal viscera is commonly associated with pulmonary tuberculosis but that the primary infection is to be searched for in the lymphatic system, especially the peribronchial and retroperitoneal glands; that the type in which tubercular peritonitis presents itself depends upon the rapidity of the inflammation, rapid processes being associated with tendency to formation of fluid and less tendency to the formation of adhesions a slower process giving rise to thickening and adhesions with fluid in the shape of walled-off collections and abscess formation, the most chronic giving rise to adhesions alone and hyperplastic growths in the walls of the intestine. Fever is usual in the acute and subacute forms and may be absent in the chronic forms, or there may be a subnormal temperature. That the fewer adhesions the better prognosis and that the ascitic form lends itself readily to operative interference. When operation is resorted to special attention should be devoted to the appendix, fallopian tubes and gall bladder.

TOXIC GASTRIC HEMORRHAGE.*

By E. L. CRISPIN, M. D., Mayo Clinic, Rochester, Minn.

While the development of gastric surgery has proved ulcer to be the most frequent cause of bleeding from the stomach and duodenum, the coincident development of pathology in the living has emphasized the fact that frequently there may be gastric hemorrhage without any demonstrable surgical gastric lesion. The opinion is prevalent among the laity that hematemesis means ulcer requiring operation, and hemorrhage from the stomach is too readily accepted by physicians as sufficient evidence to warrant surgery. I wish to call attention to the hemorrhages occurring from other than true surgical lesions, and to the importance of differentiating the causes of bleeding that are medical from those that are surgical.

The calloused ulcer derives greatest benefit from surgery. However, gastric surgery has been too often resorted to without benefit to the patient. Particularly is this true in cases in which hemorrhage was the principal cause for exploration. Often when an abnormal constitutional condition is not obvious, bleeding from the upper gastro-intestinal tract is considered as coming from a so-called hidden or non-symptomatic chronic ulcer, and the patient carries a gastro-enterostomy for ulcer for which there was not sufficient evidence before operation and no evidence at the time of operation. The burden of differentiating hemorrhage due to chronic ulcer from hemorrhage due to non-surgical conditions rests with the internist.

It is true that we occasionally see ulcers, benign and malignant, of which the histories are meager and alone are not sufficient for clinical conclusion The proportion of these will decrease with a more general knowledge of the varying clinical factors that are helpful in the recognition of the atypical group, and roentgenology will further assist in their diagnosis. Clinical study supplemented by the diagnostic efficiency developed in gastric roentgenology has made it possible to determine the presence or absence of the bleeding gastric lesions that can be benefited by surgery in a very large percentage of the cases of hemorrhage from the stomach.

To designate the oozing of blood from the stomach in the supposed absence of chronic ulcer, Sir Edwin Cooper Perry suggested to Hale White [1] the term "gastrostaxis," which is similar etymologically to "epistaxis." White [2] advanced the opinion that there might be a clinical group of this type among young women having pain, vomiting, and hematemesis, without ulcer symptoms, and in whom spontaneous recovery was the rule. The suggestion brought out considerable discussion in regard to gastric hemorrhages of obscure origin by White,[3] Bolton,[4] and Hort.[5] As the conditions in which such hemorrhages usually occur are toxic, the term "toxic gastric hemorrhage" suits our purpose better and will be used in referring to them here.

Blood that is vomited and tarry stools do not always mean hemorrhage from chronic gastric ulcer. Blood from the lungs and pharynx may be swallowed and later vomited. Bleeding from

* Read at the quarterly meeting of the Goodhue County (Minnesota) Medical Society, June 1, 1916.

esophageal varices, particularly when associated with the toxic state in cirrhosis of the liver, may be severe and have the appearance of gastric hemorrhage. In the purpuras, leukemias and anemias, especially anemias associated with enlargement of the spleen and liver, there may be severe bleeding from the stomach. In constitutional diseases and toxemias associated with hepatic and renal disease, it is common to find on necropsy that the gastric mucous membrane is intact, though vomiting of blood occurred during life. Blood may be vomited during exacerbations in states of hypertension and in secondary congestions of the liver and spleen. Endocarditis may be a remote cause of gastric hemorrhage. Also, exudative erythemic states of the viscera are possible causes of bleeding.[6] Arteriosclerosis of the abdominal vessels with aneurysmal dilatation and rupture into the stomach has been reported.[7] During the attacks of gastric crises in tabes, there is often coffee-ground vomitus and at times bleeding may be severe. Young females may have extensive hemorrhages with no proof of chronic ulcer and with usually spontaneous recovery.

With infections of the gallbladder and appendix there are occasional hemorrhages for which no adequate cause in the stomach is found at operation. In an operated series studied in the Mayo Clinic,[8] bleeding was associated with infections of the gallbladder and gastric symptoms in 5 per cent., and with appendicitis and gastric symptoms in 2 per cent. Deaver [9] mentions infections in the fallopian tubes as a causative factor in some cases of gastric hemorrhage. Bleeding from follicles or superficial erosions in the stomach permitting hemorrhage may be secondary to acute infections in the tonsils. Rosenow [10] has shown the association of various streptoccoccic infections with bleeding from hemorrhagic points and superficial ulcers in the gastric mucosa. These may become so extensive that large patchy areas of the mucosa ooze blood, though when wiped off, individual points are made out with difficulty.

Dieulafoy [11] has called attention to gastric bleeding from the two following varieties of non-chronic ulcer: (1) Simple erosions consisting of mere abrasions of the surface epithelium. These though so small as to be scarcely perceptible to the naked eye, may give rise to most alarming hemorrhages. At necropsy they may be easily overlooked, but during the course of the hemorrhage the mucous membrane appears to be studded with numerous bleeding points. (2) Exulceratio simplex. The lesions of the type to which Dieulafoy applies this term rather more extensive, and the surface layers are removed to such an extent that the arterioles running under the muscularis mucosae are exposed. This form may give rise to severe hemorrhages that may even prove fatal. On operation the condition appears as small bleeding fissures, small patchy areas oozing blood, or thick hemorrhagic infiltrations from which blood literally seeps.

Deaver,[9] in discussing hemorrhagic disease of the stomach not associated or closely related to gross ulcer, says: "Excepting extrinsic poisons, I believe the violent congestion of the gastric vessels is primarily dependent on an intra-abdominal, or more rarely, remote, focus of infection." This focus of infection he believes is most commonly the appendix or gallbladder. According to Mayo Robson [12] the gastric lesions after death in some cases of sudden severe hemorrhages, particularly in the young in whom there is no clinical evidence of ulcer, seem altogether inadequate to explain the nature of the serious hemorrhage. It is his opinion also that: "Capillary oozing or bleeding from arterioles is much more common and accounts for many more cases of gastric hemorrhage than has been hitherto supposed." Hemorrhages of this kind, parenchymatous hemorrhages in the apparently healthy male, hemorrhagic gastralgias and the large group of variously defined bleeding from the stomach in which there is sudden onset, absence of symptoms and usually spontaneous recovery, are of infective or toxic origin, and surgery will be of doubtful benefit.

Typical acute gastric ulcer may be the source of repeated hemorrhages if there is erosion of the vessels at its base, but rarely causes fatal bleeding. At operation these ulcers may be shallow and not visible or palpable through the wall of the stomach. When secondary to gross infection elsewhere, mucous ulcers may be multiple. Chronic gastric and duodenal ulcers as a rule do not bleed copiously. Bleeding from malignant disease usually is small in amount like that from ulcer, and more or less continuous.

In seeking a cause for gastric hemorrhage a history of ulcer should be sought and, when necessary, diagnostic evidence should be brought out by every adjunctive means available. If evidence indicating ulcer is not strong, effort should be made to prove or exclude all the numerous conditions that might be underlying causes of the hemorrhage.

In general, surgery offers the best results for ulcer of the chronic calloused type. In this condition the symptoms of ulcer are marked and the patient goes to the physician usually because of the distress from the ulcer rather than because of the hemorrhage. A second group of patients with gastric hemorrhage lays greater emphasis on the bleeding and complains only of gastric symptoms that are more or less indefinite. In such cases effort must be made to determine the presence or absence of calloused ulcer or of a toxic or infective condition as the cause of the hemorrhage. A third group of patients are those who come for examination because of the hemorrhage but whose gastric symptoms are of minor importance. They believe that they have an ulcer, frequently have been told that they have ulcer, and have often resorted to gastric surgery which has not prevented subsequent hemorrhages.

Speaking broadly, cases of hemorrhages without gastric symptoms, either before or after surgery, should be considered toxic. The non-surgical nature of the causative factors, such as the blood diseases and secondary congestions, are usually easy to determine. Medical observation will further develop the exact nature of many hemorrhages of the toxic group and relegate them to their proper medical sphere. When an

infective source, either intra-abdominal or remote, is found, the possibility of an association between the gastric hemorrhage and this infected atrium should be considered. In many such cases the hemorrhage is probably toxic. When medical observation determines the presence of abnormal constitutional states such as diseases of the blood, renal toxemias, disproportional varices, pathologic vascular tensions, multiple angiomas, syphilis or tuberculosis, the hemorrhage should be considered of toxic nature. Correction, when possible, of the underlying conditions, and waiting, rather than surgery, is advisable. Any surgery in these cases should be only in the nature of exploration without promise as to results. Spontaneous hemorrhages occuring in young women and parenchymatous hemorrhages in which evidence does not point to chronic ulcer, should also be considered toxic. They are not surgical; spontaneous recovery is the rule.

Patients giving a history of repeated severe hemorrhages over many years, with an ulcer history, which though not clear-cut, is strengthened by adjunctive evidence, and in whom no constitutional cause can be found, should have an exploratory operation. In such cases the hemorrhage is probably due to a surgical condition. Well-nourished patients who have had one or a few hemorrhages, and for whom the clinical or contributory data are very poor, may be considered toxic until medical observation proves the absence of a constitutional condition as a factor, or time brings out evidence of ulcer. If surgery is indicated at all for gastric hemorrhages occuring in the presence of intra or extra-abdominal infection, and accompanied by indefinite gastric symptoms, it should be applied to the focus of infection rather than to the stomach.

Patients who have had repeated hemorrhages for many years, who are past middle life, whose general appearance is below par and of whom the history and evidence of ulcer is indefinite, should also be carefully studied for toxic causes. The following abstracts of histories will serve to illustrate various types of gastric hemorrhages.

CLINICAL EVIDENCE ALONE INSUFFICIENT FOR ULCER. EXPLORATION BECAUSE OF POSITIVE ROENTGEN FINDINGS.

Case 146,581, M. C. B., traveling salesman, aged 52 years. Examined November 30, 1915. Patient had been having hemorrhages from the bowel, black and tar-like, for thirty years; in all, about twenty; in bed after each attack. The last hemorrhage occured in June, 1915. Three hemorrhages in 1913. No clear-cut gastric history, though he had had distress for days at a time, and on a few occasions for a couple of weeks. Food relief variable; never used soda; never had colic. Present trouble with stomach of about two weeks' duration. He dieted for two years but did not obtain relief. In present attack he had had a little distress at night, varying from 11 p. m. to 5 a. m., and some distress after meals. Most of the hemorrhages "have come out of a clear sky when he was feeling his best." Epistaxis frequent in youth, but never put him to bed.

Examination. A healthy looking man, 5 feet, 9 inches in height, weighing 145 pounds; no weight loss. Mucous membranes somewhat pale. Multiple pea-sized, raspberry angiomas over body. Hemoglobin 70 per cent. Gastric analysis: Acids 58, 44, 14; no food remnants. Wassermann negative. Coagulation time three minutes. Blood pressure 128, 90. Eye grounds negative. Urine negative. Protoscopic findings negative. Roentgen findings: Cap deformity; duodenal ulcer.

A history covering thirty years with slight symptoms except for bleeding, clinical absence of obstruction at the pylorus, and the angiomas over the body, made diagnosis doubtful. An exploratory operation was performed because of the roentgen evidence. A duodenal ulcer was found one-half inch below the pylorus.

CLINICAL AND ADJUNCTIVE EVIDENCE INSUFFICIENT FOR ULCER. CONSIDERED TOXIC AND NOT NOW SURGICAL.

Case 141,079; T. A. C., advertising manager of a newspaper, aged 50 years. Examined September 15, 1915. Patient had been in the habit of eating fast; he worked hard; burned the candle at both ends. Formerly a printer. Twenty-five years ago he had anemia which he thought was due possibly to lead poisoning. In the last twenty-five years he had had a few attacks of gastric trouble lasting a week or two. Symptoms meager. In October, 1914, he fainted one day while in the toilet; soon vomited food and blood. Vomited clots three times within a short period. Had tarry stools for the following three or four days. Was put on a Von Leube diet and rapidly regained his health; well since. Came for examination eleven months later because an ulcer had been diagnosed at the time of the hemorrhage. No symptoms since hemorrhage except a little discomfort without food relation, in the left hypochrondrium.

Examination. Height 5 feet, 10½ inches; weight 195 pounds; no weight loss; skin somewhat highly colored. Blood pressure 178,100. Gastric analysis: Acids 66, 56, 10. No food remnants. Hemoglobin 89 per cent. Differential blood count normal. Coagulation time eight minutes. Wasserman negative. Negative fundi. Urine showed a few hyalin casts. Roentgen finding: Stomach indeterminate.

On account of lack of evidence of ulcer, and because of the blood pressure, urine findings, coagulation time, and patient's generally well-nourished condition, diagnosis was made of toxic hemorrhage. He was advised about caring for his general health, and sent home for observation.

NEGATIVE GASTRIC EXPLORATION. CONSTITUTIONAL STATE. VISCERAL ANGIO-NEUROTIC EDEMA BELIEVED TO BE CAUSE OF BLEEDING.

Case 101,588; W. H. S., paper hanger, aged 40 years. Examined March 4, 1914. This patient had been urged a number of times to have operation for gastric ulcer. He had gastric trouble intermittently for twenty-three years. When 17 had cramps, were so severe as to double him up. He used to tie a towel tight around his waist, and a number of times was rolled over a barrel to relieve the cramping pain. These attacks came frequently for a few weeks and then disappeared for weeks or months. Only occasional trouble between the ages of 20 and 30. In an attack when about 31, he vomited a large handful of clots of blood. No clear-cut food relation to gastric pain. Present attack three weeks. No regularity. For three months had had most marked angioneurotic disturbances. Large plaques came out on his skin each night. Roentgen findings: "Lesion of the stomach at or near the pylorus." The patient had angioneurotic swellings. At exploration (C. H. Mayo) neither lesion nor cause for hemorrhage

was found in the stomach. The gallbladder, showing doubtful pathology, and the appendix were removed.

The attacks of pain have continued since operation without any material change in nature. They are regarded as visceral manifestations of angioneurotic edema. Because of the constitutional condition the hemorrhages may be considered toxic.

YOUNG WOMAN. HEMORRHAGE. TREATMENT FOR ULCER. NO SYMPTOMS OR EVIDENCE OF ULCER. SPONTANEOUS RECOVERY.

Case 147,636; Miss A. R., aged 21. Examined December 11, 1915. She complained of gastric trouble and menorrhagia. Eighteen months before a severe hemorrhage occured in the stomach; melena persisted for a number of days. She was put to bed and kept on milk diet for seven months. Her physician said the bleeding came from an ulcer in the stomach. She had been away at school, had eaten irregularly injudiciously a large amount of candy; enjoyed social activity, dancing, tennis, etc. Indefinite distress in the stomach began a month or two before the hemorrhage, about the middle of the school year. Stomach always tender; with pain and soreness after eating ordinary foods; never free periods. No food relief. Menstruation irregular, increased flow for three years; for two weeks at each period a profuse flow. Patient said she had had grippe three or four times. Wrenched her back four years ago and it "had been sore ever since."

Examination. A thin, somewhat pale young girl. Very nervous. Weight 104 pounds. Blood pressure 100, 78. Urine negative. Hemoglobin 89 per cent. Gastric analysis: Acids 20, all combined. No food remnants. Roentgen findings: Stomach negative. At the hotel the patient ate everything, and forgot all about her stomach.

Because of the hemorrhage she firmly believed that she had ulcer, as did her mother, and her brother, a physician. Her physician had advised operation for ulcer. The seven months in bed on ulcer treatment had made her a marked neurasthenic. There was neither clinical nor laboratory evidence of ulcer. Patient was told that there was no evidence of chronic ulcer and operation was not advised.

GASTRIC ATTACKS WITH HEMORRHAGES, FOLLOWING TONSILLITIS.

Case 145,217; Mrs. R. T., aged 24 years. Examined November 8, 1915. Epigastric pain, hemorrhages from stomach. She has had repeated attacks of tonsillitis and rheumatism. Distress in stomach at times for eight years. Cramp-like pains lasting five or ten minutes associated with nausea. Vomited during the first five years, but seldom vomited food. Has had numerous hemorrhages from stomach, in one of which she said she lost a quart of blood. Gastric symptoms lasted ten days to two weeks; she was then relieved for a period of months. No definite food relationship. The pain was present even when a strict diet was maintained. About three attacks of tonsillitis each winter. Gastric attacks always followed tonsillitis. In a remission four weeks before there was a hemorrhage from the stomach.

Examination. Short, soft, systolic blow heard at cardiac apex. Blood pressure 122, 82. Pulse 68. Temperature 99.2. Urine negative. Hemoglobin 70 per cent. Gastric analysis: Acids 48, 28, 20. Roentgen findings: Chest negative; stomach indeterminate.

Consultant's Note. "Not typical ulcer. History suggests superficial type of acute lesion with hemorrhage. The frequency with which tonsillitis has preceeded attacks is interesting." Tonsillectomy by Dr. Matthews. Culture from tonsils by Dr. Rosenow showed streptococci, and animals injected showed multiple hemorrhages and superficial ulcers of the stomach, due to streptococci. Hence these hemorrhages, which seemed to be toxic, were apparently due to localized hematogenous infections of the mucous membrane of the stomach, following tonsillitis.

GASTRIC SYMPTOMS WITH HEMORRHAGES. EXPLORATION: CHRONIC APPENDICITIS. NEGATIVE GALLBLADDER AND STOMACH.

Case 126,387; E. G. P., contractor and builder, aged 46 years. Examined March 12, 1915. This patient had had myalgias, followed by ecchymoses that put him in bed for three or four days. For fifteen years he had had trouble with his stomach which came on in spells formerly lasting for months. Trouble now continuous. Had had some food relief and relief by alkalies and vomiting. A burning sensation was felt in the stomach usually from 10 to 11 a. m. Hemorrhages from the stomach. Vomited a large quantity of blood during a period of two days. Four months ago had another hemorrhage and at that time tarry stools. Described pain radiation as being "most anywhere" in the upper abdomen. Patient very nervous; continuous headache for last two months.

Examination. Under weight. Blood pressure 150, 105. Hemoglobin 88 per cent. Coagulation time five minutes. Wassermann negative. Moderate right pyelitis, proved by ureteral catheterization. Gastric analysis: Acids 34, 22, 12. No food remnants. Exploration for peptic ulcer (W. J. Mayo).

Findings at operation: "Sub-acute appendicitis. Appendicitis apparently would account for symptoms as it was rather unusually well marked. Gallbladder and duodenum normal. Gallbladder somewhat adherent, but empties easily and contains no stones." Appendix removed. After negative exploration of upper abdomen, the hemorrhages were believed to be gastrotoxic, secondary to the infection in the diseased appendix.

GASTRIC SYMPTOMS, HEMORRHAGE, CAME FOR "ULCER." EXPLORATION: CHOLECYSTITIS PANCREATITIS. NEGATIVE STOMACH AND DUODENUM.

Case 124,020; Mrs. N. M., aged 36 years. Examined February 8, 1915. Trouble with stomach began four years ago. First attack, four months. One free period of three years. Second attack began one year before; was ill nine months. She had pain nearly continuously except when eating. Vomited sour water. Once she had delayed vomiting. Slight food relief. Soda relief formerly. Had sharp cutting pain in left epigastrium for which her physician had given morphia. Tarry stools. Coarse and sour foods caused distress. Patient referred to the Mayo Clinic with a diagnosis of ulcer with hemorrhage.

Examination. A very neurotic, fairly well nourished woman. Area complained of the left epigastrium. Slight tenderness to deep pressure in right lower abdomen. Blood pressure, 125, 80. Urine negative. Hemoglobin 85 per cent. White blood cells, 7000. Gastric analysis: Acids 28, 12. 16. No food remnants. Roentgen findings. Stomach indeterminate. The physician at home and the patient were sure that ulcer was the cause of her distress and bleeding. She was under observation a week, then sent for exploration of the stomach, gallbladder and the appendix. Cholecystectomy and appendectomy were done.

Findings at operation: (E. S. Judd) "Definite chronic cholecystitis and chronic pancreatitis. Head of pancreas twice its normal size, chronic appendicitis; stomach and duodenum negative."

NEGATIVE EXPLORATION—TOXIC CONSTITUTIONAL CONDITION—CARDIO RENAL SYNDROME WITH TRANSIENT HYPERTENSION.

Case 135,561; H. R. T., bank cashier, aged 60 years. Examined July 17, 1915. Patient had had hemorrhages from the bowels (tarry stools) seventeen years, ten years and five years ago. In September, 1914, also vomited blood; collapsed. In years past had to be careful of his diet and had intermittent discomfort. A clear history was difficult to obtain. A diagnosis of ulcer was made after each hemorrhage. In the Fall of 1914 a gastrojejunostomy was done for "ulcer on anterior duodenal wall one inch below the pylorus." Four weeks before coming for examination he had collapsed and the next day had profuse tarry stools.

Examination. Five feet, seven inches in height; weighed 130 pounds; underweight. Appeared weak and had marked pallor. Some bagginess under eyes. Sclera pearly; looked nephritic. Heart five inches to left. Diastolic murmur at aortic area. Blood pressure 185, 85. Hemoglobin 45 per cent. Coagulation time six minutes. Differential blood count normal. Gastric analysis: Acids 64, 54, 10. No food remnants. Wassermann negative. Old patches of hemorrhages in fundi. Phenolsulphonephthalein functional test 43 per cent. in two hours. Roentgen findings. Gastroenterostomy functioning; otherwise negative. Patient gained on ulcer diet. Was kept under observation for three weeks. Believed to be gastrotoxic, but because of reported presence of ulcer at the time of gastroenterostomy done elsewhere, and in order to clear up the nature of the condition, an exploratory operation was performed (W. J. Mayo).

Surgical report: "Two inches of the stomach and two inches of the duodenum were resected. Gastro-enterostomy in good condition. Site of supposed ulcer on duodenum resected. Patient in wretched condition." Pathologic report: Pyloric ring of stomach normal; on section scar of ulcer could not be found.

The patient gained rather slowly after operation. About three months later he had a very severe hemorrhage. Was found unconscious in a pool of blood in the bathroom. A letter from his home physician stated that a blood pressure of 250 systolic had been recorded a few days before the hemorrhage. It is probable that all of the bleedings were the result of a constitutional state, toxic in type, and of a nature that surgery could not benefit.

SUMMARY.

Toxic gastric hemorrhage is essentially a medical condition. Hemorrhage does not always mean chronic ulcer. Surgery should be resorted to for the calloused type of ulcer, and for this type only will it give the best results. Recognition of the true cause of hemorrhage from the upper gastro-intestinal tract is sometimes most difficult. At times evidence will warrant exploration to prove or exclude peptic ulcer as a cause. The presence of a constitutional disease without sufficient evidence of ulcer, makes medical observation and study, rather than surgery, advisable. In cases of hemorrhages of obscure origin, search for infected foci should be made and the possibility of their association with the cause of the hemorrhage should be considered. In addition, studies of blood diseases associated with bleeding, and further studies in blood pressure, with recognition of transient hypertensive states, will help to define and separate hemorrhages having their origin in surgical ulcer from gastric hemorrhages of acute infective and toxic origins.

References.

1. White, W. H.—Gastrostaxis or Oozing of Blood from the Mucous Membrane of the Stomach. Lancet, 1906, ii, 1189-95.
2. White, W. H.—Are Not Some Patients Said to be Afflicted with Gastric Ulcer Really Suffering from a Different Disease? Lancet, 1901, i, 1819-20.
3. White, W. H.—Gastrostaxis. Brit. Med. Jour., 1910, i, 1347-8.
4. Bolton, C.—Does Gastrostaxis Exist as an Independent Disease? Brit. Med. Jour., 1910, i, 1221-24.
5. Hort, E. C.—Gastrostaxis. Brit. Med. Jour., 1910, i, 1443.
6. Crispin, E. L.—Visceral Crises in Angioneurotic Edema. St. Paul Med. Jour., 1916, xviii, 127-33.
7. Hood, D. W. C.—Hematemesis Due to Arteriosclerosis of the Gastric Vessels. Lancet, 1912, ii, 1177.
8. Eusterman, G. B.—The Essential Factors in the Diagnosis of Chronic and Duodenal Ulcers. Jour. Amer. Med. Assn., 1915, lxv, 1500-03.
9. Deaver, J. B.—Gastric Hemorrhage. Surg., Gynec. and Obst., 1914, xviii, 294-99.
10. Rosenow, E. C.—Elective Localization of Streptococci. Jour. Amer. Med. Assn., 1915, lxv, 1687-91.
11. Dieulafoy, quoted by Robson, A. W. M.—Discussion on the Surgical Treatment of Hematemesis. Lancet, 1902, ii, 1626-30.
12. Robson, A. W. M.—Discussion on the Surgical Treatment of Hematemesis. Lancet, 1902, ii, 1626-30.

HYPOPHYSEAL SYMPTOMATOLOGY; A REVIEW.*

By CARL WHEELER RAND, M. A., M. D., Los Angeles.

Recent conceptions of the ductless glands differ from previous ideas, chiefly in that observers are now beginning to recognize the inter-dependence of these organs; to consider them more as inseparable complexes; and are striving to estimate correctly both their individual and reciprocal influences. The clinical manifestations resulting from endo-secretory disorders are usually described as composing one or more phases of a general polyglandular syndrome. It is the purpose of this paper to consider, very briefly, certain influences which the pituitary body may play in the production of some of these symptom-complexes.

In 1838, Rathke [22] described an ectodermic pouch budding from the bucco-pharangeal cavity which meets and partly surrounds the infundibular prolongation of the anterior cerebral vesicle. The tip of this prolongation subsequently becoming thickened, is known as the infundibular body, neurohypophysis or pars nervosa. Later the developing sphenoid bone obliterates the lumen of Rathke's pouch, the tip of which envelopes the infundibular body. This combined neuro-epithelial structure, or pituitary body proper, later becomes endowed with a dural capsule and occupies the sella turcica. It is present in all vertebrates showing its phylogenetic importance.

Histologically, the gland is divided into three parts—(I) the anterior lobe (pars anterior, pituitary gland proper) consisting of columns of cells surrounded by large sinusoidal spaces. These cells are classified by Flesch [10] (1884) into so-called chromo-philes, composed of eosino-philes and basophiles; and chromo-phobes or neutro-philes. These cells show hypertrophic changes during pregnancy; atrophic during hibernation, etc.; (II) the posterior lobe (pars nervosa, infundibular body, neuro hypophyses, etc.) composed of a meshwork of loosely placed neurologia whose fibres radiate toward the infundibulum; (III) the pars intermedia of Herring, or the epithelial investment of the infundibulum. The two become fused. Structur-

* Read before the Southern California Medical Society at Pomona, California, May 4, 1916.